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# Effect of topical brinzolamide 1% and brimonidine 0.2% on intraocular pressure after phacoemulsification

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**Purpose:** To compare the effectiveness of brinzolamide 1% (Azopt®) and brimonidine 0.2% (Alphagan®) with a placebo in preventing an early increase in intraocular pressure (IOP) after phacoemulsification.

**Setting:** Department of Ophthalmology, Baskent University, School of Medicine, Ankara, Turkey.

**Methods:** In this prospective double-masked study, 90 eyes of 90 patients having clear corneal phacoemulsification were randomly divided into 3 groups of 30 eyes each. One hour before surgery, 1 group received 1 drop of brinzolamide 1%, another received 1 drop of brimonidine 0.2%, and the third received 1 drop of a balanced saline solution (placebo). The IOP was measured preoperatively and 3 and 16 to 20 hours postoperatively.

**Results:** Three hours postoperatively, the mean IOP increased by 4.2 mm Hg  $\pm$  7.0 (SD), 3.2  $\pm$  6.4 mm Hg, and 5.3  $\pm$  4.2 mm Hg in the brinzolamide, brimonidine, and placebo groups, respectively. The IOP increase from baseline was significant in all 3 groups (all  $P < .01$ ), with no difference between the groups ( $P > .05$ ). The change in IOP at 16 to 20 hours was 0.2  $\pm$  2.8 mm Hg, 0.2  $\pm$  2.4 mm Hg, and  $-0.8 \pm 2.4$  mm Hg, respectively. The changes were not significant compared to baseline (all  $P > .05$ ). Six eyes (20%) in the brinzolamide group, 5 eyes (16.7%) in the brimonidine group, and 7 eyes (23.3%) in the placebo group had an IOP higher than 25 mm Hg 3 hours postoperatively; the difference between groups was not significant ( $P = .8$ ).

**Conclusion:** Prophylactic use of 1 drop of brinzolamide or brimonidine was not more effective than a placebo in controlling early postoperative IOP elevations after clear corneal phacoemulsification.

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Modern cataract surgery with phacoemulsification and implantation of a foldable intraocular lens (IOL) has many advantages. The main benefits are

more rapid wound healing, faster visual recovery and refractive outcomes, and greater patient satisfaction. These benefits allow many patients to be discharged soon after surgery and the first postoperative evaluation performed at 1 day or later. However, an early postoperative increase in intraocular pressure (IOP) is a serious, well-documented event that surgeons should consider in every case. This rise typically occurs during the first 24 hours after cataract surgery<sup>1–5</sup> and is most marked at 3 to 7 hours.<sup>6,7</sup> The spikes can be dangerous in eyes with optic discs that are vulnerable as a result of preexisting damage from glaucoma or atherosclerosis-induced blood flow compromise. Corneal edema and

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postoperative ocular pain are the other complications of postoperative IOP spikes.

Many strategies to prevent or minimize these IOP rises have been tried including surgical techniques for removing viscoelastic material,<sup>4,8</sup> prophylactic use of intracameral cholinergic agents,<sup>8,9</sup> and use of topical or systemic antiglaucoma agents.<sup>8-15</sup> Studies report good results with intracameral acetylcholine,<sup>8</sup> timolol gel,<sup>10,13</sup> a dorzolamide-timolol combination,<sup>11</sup> dorzolamide alone,<sup>15</sup> acetazolamide,<sup>16</sup> and pilocarpine gel<sup>17</sup>; however, an optimally effective, safe, and rapid-onset prophylactic agent that lasts for 24 hours and results in a predictable, equal effect in every case is not available. Such an agent is particularly needed in eyes with preexisting glaucoma.

Brinzolamide 1% is a carbonic anhydrase inhibitor that acts as an aqueous suppressant. It begins to act 30 minutes after administration, shows peak effect at 2 hours, and lasts for 6 to 8 hours. To our knowledge, there is no published research of the effect of brinzolamide on IOP spikes after cataract surgery.

Brimonidine 0.2%, an  $\alpha_2$ -agonist, is also an aqueous suppressant; however, it acts by increasing uveoscleral, and possibly trabecular, outflow. This agent starts to act within 1 hour of administration, shows peak effect at 2 to 3 hours, and lasts for 10 to 14 hours. Brimonidine 0.2% effectively prevents IOP rises after neodymium:YAG laser peripheral iridectomy and posterior capsulotomy<sup>18,19</sup> and after argon laser trabeculoplasty.<sup>18,20</sup>

We conducted a prospective randomized placebo-controlled double-masked study of the IOP-lowering effects of brinzolamide 1% and brimonidine 0.2% after phacoemulsification. Each drug was administered approximately 1 hour before surgery, and the IOP effects were assessed twice within 24 hours after surgery.

## Patients and Methods

This study comprised 90 eyes of 90 consecutive patients who had phacoemulsification cataract surgery with foldable IOL implantation. All the surgeries were performed between May and June 2002 by 1 of 2 surgeons (A.A., Y.A.A.) using the same technique. All patients provided informed consent. The study design was approved by the Ethical Committee of Baskent University, and the study followed the tenets of the Declaration of Helsinki.

The exclusion criteria were previous ocular surgery or laser treatment involving angle structures, ocular hypertension (IOP greater than 21 mm Hg), pigment dispersion syndrome, exfoliation syndrome, hypersensitivity to sulfon-

amides, any form of glaucoma, and complications during cataract surgery.

A computer-generated randomization list was used to assign patients to 1 of 3 groups of 30 each based on what preoperative eyedrop they were to receive: brinzolamide 1% (Azopt®), brimonidine 0.2% (Alphagan®), or a balanced saline solution (placebo). All patients received 1 drop of the respective agent approximately 1 hour before surgery. The surgeon was masked to the treatment group assignments.

### *Surgical Technique*

All eyes received phenylephrine 2.5%, cyclopentolate 1%, and tropicamide 0.5% approximately 1 hour before surgery to dilate the pupil. After peribulbar anesthesia of articaine hydrochloride 2% and bupivacaine hydrochloride 0.5% was administered, a 3.0 mm, clear corneal, self-sealing incision was made at the 11 o'clock position and the anterior chamber was filled with sodium hyaluronate 3%-chondroitin sulfate 4% (Viscoat®). Phacoemulsification was by a standard phaco-chop technique. After phacoemulsification and cortical cleanup, the capsular bag was expanded using sodium hyaluronate 1% (Biolon®), 10 mg/mL. Next, a single-piece, 6.0 mm, hydrophobic, foldable acrylic IOL (AcrySof®, MA60BM, Alcon) was implanted in the bag.

An irrigation/aspiration (I/A) cannula was used with the rock 'n roll technique to aspirate remaining viscoelastic material from the prelental and retrolental spaces, capsule fornix, and anterior chamber for at least 30 seconds until no viscoelastic material was visible under the operating microscope. In the rock 'n roll technique, the I/A tip is placed on the IOL surface. The I/A cannula is rolled back and forth, and intermittent gentle pressure is applied to mobilize the viscoelastic material trapped between the IOL and the capsular bag during I/A.

The incision was left sutureless, and no miotics were used intracamerally at the end of surgery. Subconjunctival injections of cefazolin sodium (50 mg/mL) and dexamethasone phosphate (8 mg/mL) were administered in the operated eye. Right after surgery, the IOP was measured with a sterile-tipped handheld tonometer (Tono-Pen, Mentor Inc.). The IOP was adjusted to a target of  $10 \pm 3$  mm Hg by exchanging balanced saline solution through the paracentesis puncture just before the eye was patched with tobramycin 0.3% ointment.

A Goldmann applanation tonometer was used to measure IOP 1 day before surgery (baseline), 3 hours postoperatively, and 16 to 20 hours postoperatively. The same examiner who was masked to the group assignments recorded all measurements using the same equipment for each measurement.

### *Statistical Analysis*

All variables were tested for normality. A 1-way analysis of variance (ANOVA) and repeated-measures ANOVA were used to analyze intragroup and intergroup IOP differences.

**Table 1.** Demographics of patients by study group.

Characteristic	Group		
	Brinzolamide	Brimonidine	Placebo
Eyes, n	30	30	30
Mean age, y	64.6 ± 8.6	67.7 ± 8.2	66.6 ± 8.5
Age range, y	48–80	50–82	56–80
Sex, M:F	11:19	14:16	12:18

Means ± SDs

The Tukey HSD test was used for post-hoc analysis. Categorized data were analyzed by the chi-square test. A *P* value less than 0.05 was considered significant.

Sample-size calculations were based on detecting a minimum 2 mm Hg difference in mean IOP in the 3 groups with an equal number of patients at 3 hours and 16 to 20 hours postoperatively. The study had 78% power to detect a 2 mm Hg IOP difference among the 3 study groups (minimum 30 patients per group) at 3 hours assuming a standard deviation of ±5 (*P* = .05, 2 tailed). The power of the test to detect a 1 mm Hg difference in IOP remained 78% 16 to 20 hours postoperatively assuming a standard deviation of ±2.5 (*P* = .05, 2 tailed).

Statistical analyses were performed using SPSS for Windows, version 9.0 (SPSS Inc.). Power analysis and sample-size calculations were performed using PASS 2002 (NCSS Statistical Software).

## Results

The mean age of the 53 women and 37 men was 66.9 years ± 7.8 (SD) (range 48 to 82 years). Table 1 shows the patients' demographics by study groups.

Table 2 shows the IOP at each time point and the changes from baseline. The mean preoperative IOP was not statistically significantly different between groups (*P* > .05).

The IOP 3 hours postoperatively was statistically significantly higher than at baseline in all groups (brinzolamide, *P* = .003; brimonidine, *P* = .01; placebo,

**Table 3.** The number of eyes with an IOP elevation greater than 5 mm Hg and 10 mm Hg 3 hours postoperatively.

Group	IOP Rise	
	>5 mm Hg	>10 mm Hg
	Eyes (%)	Eyes (%)
Brinzolamide	14 (46.7)	5 (16.7)
Brimonidine	14 (46.7)	2 (6.7)
Placebo	15 (50.0)	4 (13.3)

*P* = .001). However, the difference in IOP at 3 hours between groups was not statistically significant (*P* > .05).

At 16 to 24 hours, the mean IOP was not significantly different from baseline in any group. In addition, there were no statistically significant differences between groups (all *P* > .05).

Table 3 shows the number of eyes in each group that had an increase of 5 mm Hg and 10 mm Hg over baseline 3 hours postoperatively. There was no between-group difference in the number of eyes with a greater than 5 mm Hg increase in IOP at 3 hours (*P* > .05). Three eyes (10%) in the brinzolamide group, 1 eye (3.4%) in the brimonidine group, and no eye in the placebo group had an IOP increase greater than 5 mm Hg at 16 to 20 hours. No eye in any group had an IOP increase greater than 10 mm Hg at 16 to 20 hours.

At 3 hours, 6 eyes (20%) in the brinzolamide group, 5 eyes (16.7%) in the brimonidine group, and 7 eyes (23.3%) in the placebo group had an IOP higher than 25 mm Hg. The highest postoperative IOP was 34 mm Hg in the brinzolamide group, 31 mm Hg in the brimonidine group, and 30 mm Hg in the placebo group. There was no statistically significant difference between the 3 groups in the incidence of IOP over 25 mm Hg ( $\chi^2 = 0.417$ ; degrees of freedom = 2; *P* = .8). At 16 to 20 hours, no eye in any group had an IOP greater than 25 mm Hg.

**Table 2.** Mean IOP values (mm Hg) over time and mean change from baseline.

Group	Baseline IOP	Mean ± SD			
		3 Hours Postop		16–20 Hours Postop	
		IOP	Change from Baseline	IOP	Change from Baseline
Brinzolamide	15.5 ± 2.2	19.7 ± 6.1	4.2 ± 7.0	15.7 ± 2.6	0.2 ± 2.8
Brimonidine	15.7 ± 2.7	19.0 ± 5.4	3.2 ± 6.4	16.0 ± 2.7	0.2 ± 2.4
Placebo	15.6 ± 1.8	20.9 ± 5.0	5.3 ± 4.2	14.8 ± 2.6	−0.8 ± 2.4

No patient had a drug-related adverse event during the study period.

## Discussion

There is no agreement in the literature on the mechanism for IOP elevation after cataract surgery. This complication used to be called Healon-block glaucoma<sup>21,22</sup> when the reason was thought to be disturbed outflow facility caused by obstruction of trabeculum with this high-molecular-weight viscoelastic substance. This theory was supported by a study that reported a 32% decrease in outflow facility when an attempt was made to clear the viscoelastic material from the anterior chamber; however, a 65% decrease was noted when the viscoelastic material was left in the anterior chamber.<sup>23</sup> Theoretically, it was believed that low-molecular-weight (less-viscous) substances would leave the anterior chamber quicker and increase postoperative IOP less. Schubert and coauthors<sup>24</sup> showed that less-viscous sodium hyaluronate resulted in higher levels of more prolonged IOP rise in monkey eyes than more-viscous sodium hyaluronate.

Later reports proposed causes such as mechanical deformation of angle structures before or during surgery, inflammation, hemorrhage, pigment dispersion, and retained lens material. They theorize that these factors, together with retained viscoelastic material, cause mechanical obstruction of outflow channels, resulting in postoperative IOP elevation.<sup>7,23,25-27</sup>

Whatever the cause, many reports recommend dilution, clearance, and/or aspiration of viscoelastic material from the anterior chamber at the end of the phacoemulsification to prevent postoperative IOP elevation.<sup>21,28-30</sup> However, many others stress that this kind of intervention would not prevent the IOP rise but only decrease its level or shorten its duration to some extent.<sup>28,30</sup> Research shows that high-molecular-weight viscoelastic substances exit the eye in an unmetabolized state via the outflow channels and that these materials are completely gone by about 24 hours after cataract surgery.<sup>31,32</sup> It has also been observed that IOP normalizes toward the end of this first 24 hours.<sup>23,25,27</sup>

It is now believed that the reason IOP rises after phacoemulsification is not limited to residual viscoelastic material but also depends on the patient's characteristics and the amount of trauma during surgery. Two of

these steps—minimizing trauma during surgery and removing as much viscoelastic material as possible from the anterior chamber after IOL implantation—can be done in all cases. As Tanaka and coauthors<sup>26</sup> recommend, the surgeons in our study spent at least 30 seconds clearing the anterior chamber of viscoelastic material. Aspiration lasted until no material was visible under the operating microscope. Also, IOP was adjusted to  $10 \pm 3$  mm Hg immediately after surgery to standardize the groups. We believe that to prevent bias when comparing IOP spikes between groups, it is important that all groups begin at the same IOP level.

In our study, the mean IOP increase in the brinzolamide and brimonidine groups 3 hours after surgery was  $4.2 \pm 7.0$  mm Hg and  $3.2 \pm 6.4$  mm Hg, respectively, which was not significantly different from the mean elevation in the placebo group. Intraocular pressure spikes higher than 25 mm Hg occurred in almost 20% of eyes in all 3 study groups, with no between-group difference in the frequency. Thus, neither brinzolamide nor brimonidine effectively prevented an IOP rise or potentially dangerous IOP spikes after phacoemulsification when administered 45 to 60 minutes before surgery. By 16 to 20 hours after surgery, the mean IOP in all 3 groups had returned to baseline level.

In contrast to previous investigations, we administered the prophylactic agents preoperatively. In most studies in the literature,<sup>11-13,15</sup> topical prophylactic agents were applied immediately after surgery. The exception is 1 study<sup>14</sup> in which latanoprost was given 2 hours before phacoemulsification; it had no postoperative IOP-lowering effect. In our investigation, we instilled the medication approximately 45 to 60 minutes before surgery, when the aqueous dynamics and integrity of the anterior chamber structures were not disturbed by surgery. As described, the main action of both topical antiglaucoma agents in our study is aqueous suppression. Like all aqueous suppressants, brinzolamide and brimonidine are most effective under optimum aqueous production. When aqueous dynamics change or aqueous production is affected by surgery, it is reasonable to speculate that aqueous suppressants may not perform optimally. In addition, the documented pharmacologic properties of these agents (eg, onset of action, duration of action, peak efficacy) relate to physiologic conditions in which aqueous production rate, outflow facility, and pH of the media are normal. When these standardized

conditions are altered by surgery, the onset or duration of the drug's action changes. We administered the drugs before surgery to make use of their known properties and effectiveness.

Carbonic anhydrase inhibitors other than brinzolamide (ie, dorzolamide and acetazolamide) prevent IOP spikes to some extent after cataract surgery.<sup>10,15,16</sup> We believe our investigation is the first to indicate that brinzolamide is not effective in preventing IOP spikes after phacoemulsification. The reason is unclear. Brinzolamide has a relatively short duration of action, which might explain its lack of effectiveness. However, the IOP 3 hours after surgery (roughly 4 hours after administration) was well within the documented duration of action of this drug. Perhaps this agent does not affect outflow facility, which is thought to be the main mechanism in IOP spikes.

Brimonidine 0.2% is frequently used to treat glaucoma and ocular hypertension. Several studies show this drug effectively prevents IOP spikes after anterior segment laser surgery.<sup>18-20</sup> In 1 study,<sup>33</sup> apraclonidine 1%, another relatively selective  $\alpha_2$ -agonist, prevented IOP increases 6 hours after phacoemulsification compared to a control group when instilled 30 minutes and immediately postoperatively. The efficacy of apraclonidine 1% in decreasing large IOP elevations in glaucomatous eyes having combined extracapsular cataract extraction and trabeculectomy has also been reported.<sup>34</sup> However, 2 studies report that apraclonidine does not prevent a postoperative IOP increase after cataract surgery.<sup>35,36</sup> One study, in which brimonidine was administered immediately after small-incision cataract surgery, found the drug did not effectively prevent IOP spikes 6 hours and 24 hours postoperatively.<sup>12</sup> To date, no study has assessed whether preoperative brimonidine prevents early IOP spikes after phacoemulsification. However, we administered phenylephrine (an  $\alpha$ -receptor agonist) for pupil dilation and brimonidine (another  $\alpha$ -receptor agonist) at the same time, 1 hour before surgery. This may explain why brimonidine did not prevent postoperative IOP spikes, as hypothesized in another study.<sup>12</sup> Miotics, which increase the outflow facility, have been reported to effectively prevent postoperative IOP rises after phacoemulsification.<sup>8,17</sup> Another reason for the ineffectiveness of brimonidine and brinzolamide in our study may be because these drugs do not increase the outflow facility in a classic manner.

We found no long-lasting pressure rise in the 3 groups throughout the study period. The highest postoperative IOP in the 90 eyes in the study was 34 mm Hg. Although this level is dangerous in patients with glaucoma or a disc vulnerable to damage by high IOP, if transient, it is acceptable in eyes with a healthy optic disc. Thus, prophylactic IOP-lowering therapy is not needed in all patients having phacoemulsification, only in those with a previously damaged optic disc.

In conclusion, it appears that neither brinzolamide nor brimonidine administered 45 to 60 minutes before surgery prevents the early IOP rise that typically occurs after phacoemulsification.

## References

1. Rowes JR. Postoperative IOP in cataract surgery [letter]. *Ophthalmic Surg* 1989; 20:145
2. Kooner KS, Cooksey JC, Perry P, Zimmerman TJ. Intraocular pressure following ECCE, phacoemulsification, and PC-IOL implantation. *Ophthalmic Surg* 1988; 19: 643-646
3. Fry LL. Postoperative intraocular pressure rises: a comparison of Healon, Amvisc, and Viscoat. *J Cataract Refract Surg* 1989; 15:415-420
4. Kohnen T, von Ehr M, Schütt E, Koch DD. Evaluation of intraocular pressure with Healon and Healon GV in sutureless cataract surgery with foldable lens implantation. *J Cataract Refract Surg* 1996; 22:227-237
5. Ruiz RS, Wilson CA, Musgrove KH, Prager TC. Management of increased intraocular pressure after cataract extraction. *Am J Ophthalmol* 1987; 103:487-491
6. Ahmed IIK, Kranemann C, Chipman C, Malam F. Revisiting early postoperative follow-up after phacoemulsification. *J Cataract Refract Surg* 2002; 28:100-108
7. Krug JH Jr. Glaucoma after cataract surgery. In: Albert DM, Jakobiec FA, eds, *Principles and Practice of Ophthalmology*, 2nd ed. Philadelphia, PA, WB Saunders, 2000; 2824-2834
8. Wedrich A, Menapace R. Intraocular pressure following small-incision cataract surgery and polyHEMA posterior chamber lens implantation; a comparison between acetylcholine and carbachol. *J Cataract Refract Surg* 1992; 18:500-505
9. Fry LL. Comparison of the postoperative intraocular pressure with Betagan, Betoptic, Timoptic, Iopidine, Diamox, Pilopine Gel, and Miostat. *J Cataract Refract Surg* 1992; 18:14-19
10. Kanellopoulos AJ, Perry HD, Donnenfeld ED. Timolol gel versus acetazolamide in the prophylaxis of ocular hypertension after phacoemulsification. *J Cataract Refract Surg* 1997; 23:1070-1074

11. Rainer G, Menapace R, Findl O, et al. Intraindividual comparison of the effects of a fixed dorzolamide-timolol combination and latanoprost on intraocular pressure after small incision cataract surgery. *J Cataract Refract Surg* 2001; 27:706-710
12. Rainer G, Menapace R, Findl O, et al. Effect of topical brimonidine on intraocular pressure after small incision cataract surgery. *J Cataract Refract Surg* 2001; 27:1227-1231
13. Lai JSM, Chua JKH, Leung ATS, Lam DSC. Latanoprost versus timolol gel to prevent ocular hypertension after phacoemulsification and intraocular lens implantation. *J Cataract Refract Surg* 2000; 26:386-391
14. Lai JSM, Loo A, Tham CCY, et al. Preoperative latanoprost to prevent ocular hypertension after phacoemulsification and intraocular lens implantation. *J Cataract Refract Surg* 2001; 27:1792-1795
15. Rainer G, Menapace R, Findl O, et al. Randomised fellow eye comparison of the effectiveness of dorzolamide and apraclonidine on intraocular pressure following phacoemulsification cataract surgery. *Eye* 2000; 14:757-760
16. Lewen R, Insler MS. The effect of prophylactic acetazolamide on the intraocular pressure rise associated with Healon-aided intraocular lens surgery. *Ann Ophthalmol* 1985; 17:315-318
17. Ruiz RS, Wilson CA, Musgrove KH, Prager TC. Management of increased intraocular pressure after cataract extraction. *Am J Ophthalmol* 1987; 103:487-491
18. Chevrier RL, Assalian A, Duperré J, Lesk MR. Apraclonidine 0.5% versus brimonidine 0.2% for the control of intraocular pressure elevation following anterior segment laser procedures. *Ophthalmic Surg Lasers* 1999; 30:199-204
19. Gartaganis SP, Mela EK, Katsimpris JM, et al. Use of topical brimonidine to prevent intraocular pressure elevations following Nd:YAG-laser posterior capsulotomy. *Ophthalmic Surg Lasers* 1999; 30:647-652
20. Barnes SD, Campagna JA, Dirks MS, Doe EA. Control of intraocular pressure elevations after argon laser trabeculoplasty; comparison of brimonidine 0.2% to apraclonidine 1.0%. *Ophthalmology* 1999; 106:2033-2037
21. Barron BA, Busin M, Page C, et al. Comparison of the effects of Viscoat and Healon on postoperative intraocular pressure. *Am J Ophthalmol* 1985; 100:377-384
22. Hoffer KJ. Effects of extracapsular implant techniques on endothelial density. *Arch Ophthalmol* 1982; 100:791-792
23. Berson FG, Patterson MM, Epstein DL. Obstruction of aqueous outflow by sodium hyaluronate in enucleated human eyes. *Am J Ophthalmol* 1983; 95:668-672
24. Schubert HD, Denlinger JL, Balazs EA. Exogenous N-hyaluronate in the anterior chamber of the owl monkey and its effect on the intraocular pressure. *Exp Eye Res* 1984; 39:137-152
25. Passo MS, Ernest JT, Goldstick TK. Hyaluronate increases intraocular pressure when used in cataract extraction. *Br J Ophthalmol* 1985; 69:572-575
26. Tanaka T, Inoue H, Kudo S, Ogawa T. Relationship between postoperative intraocular pressure elevation and residual sodium hyaluronate following phacoemulsification and aspiration. *J Cataract Refract Surg* 1997; 23:284-288
27. Naeser K, Thim K, Hansen TE, et al. Intraocular pressure in the first days after implantation of posterior chamber lenses with the use of sodium hyaluronate (Healon®). *Acta Ophthalmol* 1986; 64:330-337
28. Glasser DB, Matsuda M, Edelhofer HF. A comparison of the efficacy and toxicity of and intraocular pressure response to viscous solutions in the anterior chamber. *Arch Ophthalmol* 1986; 104:1819-1824
29. Miller D, Stegmann R. The use of Healon in intraocular lens implantation. *Int Ophthalmol Clin* 1982; 22(2):177-187
30. Olivius E, Thorburn W. Intraocular pressure after cataract surgery with Healon®. *Am Intra-Ocular Implant Soc J* 1985; 11:480-482
31. Balazs EA. Sodium hyaluronate in viscosurgery. In: Miller D, Stegmann R, eds, *Healon (Sodium Hyaluronate); a Guide to Its Use in Ophthalmic Surgery*. New York, NY, John Wiley, 1983; 5-28
32. Morgan RK, Skuta GL. Viscoelastic-related glaucomas. *Semin Ophthalmol* 1994; 9:229-234
33. Araie M, Ishi K. Effects of apraclonidine on intraocular pressure and blood-aqueous barrier permeability after phacoemulsification and intraocular lens implantation. *Am J Ophthalmol* 1993; 116:67-71
34. Robin AL. Effect of topical apraclonidine on the frequency of intraocular pressure elevations after combined extracapsular cataract extraction and trabeculectomy. *Ophthalmology* 1993; 100:628-633
35. Byrd S, Singh K. Medical control of intraocular pressure after cataract surgery. *J Cataract Refract Surg* 1998; 24:1493-1497
36. Bömer TG, Lagrèze W-DA, Funk J. Intraocular pressure rise after phacoemulsification with posterior chamber lens implantation: Effect of prophylactic medication, wound closure, and surgeon's experience. *Br J Ophthalmol* 1995; 79:809-813